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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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EXAMINER	
HADDAD, MAHER M	

ART UNIT	PAPER NUMBER
1644	

NOTIFICATION DATE	DELIVERY MODE
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Notice of the Office communication was sent electronically on above-indicated "Notification Date" to the following e-mail address(es):

mailroom@bskb.com

Office Action Summary	Application No.	Applicant(s)	
	10/526,372	OHIZUMI ET AL.	
	Examiner	Art Unit	
	Maher M. Haddad	1644	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *04 June 2009*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,2,5-7,9,13-18,21,22 and 24* is/are pending in the application.

4a) Of the above claim(s) *15,17,22 and 24* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-2, 5-7, 9, 13-14, 16 and 18-21* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

RESPONSE TO APPLICANT'S AMENDMENT

1. Applicant's amendment, filed 6/4/09, is acknowledged.

2. Claims 1-2, 5-7, 9, 13-18, 21-22 and 24 are pending.

3. Claims 15, 17, 22 and 24 stand withdrawn from further consideration by the Examiner, 37 C.F.R. § 1.142(b) as being drawn to a nonelected invention.

4. Claims 1-2, 5-7, 9, 13-14, 16 and 18-21 are under consideration in the instant application.

5. In view of the amendment filed on 6/4/09, only the following rejections are remained.

6. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless --

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

7. Claims 7, 9, 14, 16 and 18 stand rejected under 35 U.S.C. 102(b) as being anticipated by U.S. Pat. No. 6,235,714 for the same reasons set forth in the previous Office Action mailed 12/05/08.

Applicant's arguments, filed 6/4/09, have been fully considered, but have not been found convincing.

Applicant submits that claim 7 recites a human native protein which does not read on the referenced PKKKMEK peptide. Further Applicant argues that the `714 patent does not describe the use of soluble antigens. Applicant argues that the `714 patent teaches that "if high levels Ab titers are not reached [after the immunization of A431 cell], booster injections wit the soluble extracellular domain of the epidermal growth factor receptor (exEGFR) will be administered," (col., 14, lines 52-54). Applicant concluded that the `714 patent teaches that the immunization of A431 cells does not elicit antibody. The `714 patent simply does not describe whether or not a desired antibody can be obtained by immunization with exEGFR. Instead, the `714 patent teaches that TSA-EGFR, conjugated with KLH, is immunized to a MRL/lpr mouse, such that the immune system generates a catalytic antibody. That is, the `714 patent merely discloses that TSA-EGFR should be use to generate catalytic antibodies. Applicant submits that that `714 patent does not demonstrate that the immunization of the human native protein, EGFR or exEGFR, elicits an antibody. Moreover, the percentage of sequence identity of EGFR or exEGFR between a human and a mouse is 78%. The `714 patent does not teach or suggest that antibodies can be generated using an immunogen, which shareds a sequence identity between mouse and human greater than of EGFR or exEGFR.

However, the claimed invention encompasses producing any antibodies whether the antibodies are catalytic or non-catalytic antibodies. Regarding the issue that the `714 patent does not demonstrate the immunization with EGFR or exEGFR, elicits an antibody, the Examiner notes that the `714 patent teaches the use of EGFR or exEGFR to produce antibodies using the MRL/lpr mouse (see fig. 19 and col., 8, under selection and preparation of CRAAs in particular). Regarding the fact that the human EGFR is 78% sequence identical to mouse, the examiner notes that the reference is not limited to human EGFR protein. The `714 patent teaches other target antigens listed in Fig. 19 such as Macrophage inhibitory factor (MIF), C5, GPIIb/IIIa receptor (96% at the amino acid level), FVII, IL-4, IL-5, IgE, Eotaxin, PDGF, αvβ3 integrin (96% at the amino acid level). Applicant fails to address the homology of said antigens. These antigen protein exhibits high amino acid sequence homology in a human and mouse, wherein the native protein has a sequence identity more than 94% at the "amino acid sequence level" to a homolog protein of the nonhuman animal to be immunized in the absence of evidence to the contrary.

8. The following new ground of rejections are necessitated by Applicant's amendment filed 6/4/09.

9. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> *(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.*

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(f) or (g) prior art under 35 U.S.C. 103(a).

10. Claims 1-2, 5-7, 9, 13-14, 16 and 18-21 are rejected under 35 U.S.C. 103(a) as being unpatentable over JP-01047390 (IDS ref. No. BB) OR US. Pat. No. 4,965,198 (IDS AA), each in view of Makino et al (J Clin Lab Immunol. 1988 Feb;25(2):83-8) and Lage et al (2001, IDS CA).

The `390 publication teaches that a mouse having an autoimmune disease such as MRL/I mouse can be used to produce a monoclonal antibody (see the English translation provided by Applicant). The `390 publication teaches a method of producing a hybridoma which produces the monoclonal antibody, wherein an animal having an autoimmune disease is used as a mammal from which plasma cells are obtained. It is preferable that the animal is selected considering the adaptability to myeloma used for cell fusion. A mouse or rat is preferable, wherein the mouse having an autoimmune disease includes N2B, NZW, B/WF1, MRL/I, BXSB male and SLN1 strain. A rat having an autoimmune disease includes a rat in which hypertension occurs spontaneously. Further, a normal mouse such as Balb/c of which the ability to produce

autoantibodies increases by being administered with a polyclonal B cells activator such as lipopolysaccharide (LPS) of a gram negative bacterium and dextran sulfate and which is in the state of autoimmune disease may be used (see the Partial English translation of Japanese Publication No. 0104739).

The `198 patent teaches a preparation containing immunogens is used to immunize animals. Thereupon, the immunized animals are preferred to be selected with consideration of their compatibility with the myeloma used in cell fusion. Mice or rats are preferable. When using glycolipids which contain N-glycolylneuraminic acid, an object of this invention, animals with autoimmune disease are more preferable and mice with autoimmune disease are the most preferable. As the mice with autoimmune disease, there are NZB, NZW, B/WF1, MRL/l, BXSB (SLE) male, SL/Ni and other mice available. Normal mice such as Balb/c may be used as immunized animals if the mice become autoimmune by raising their autoantibody producing ability caused by the injection of a polyclonal B cell activator (PBA) such as bacterial lipopoly-saccharide (LPS) or dexstran sulfate (col. 8, lines 37-53 and claim 12 in particular). The `198 patent teaches that the glycolipids which contain N-glycolylneuraminic acid including gangliosides with the H-D antigen activity, one of the objects in this invention, are known to exist widely in mouse tissues, so that these glycolipids are autoantigens for mice. Therefore, these glycolipids are thought to have extremely weak immunogenicity. It is very difficult to obtain the monoclonal antibody specific to or against glycolipids containing N-glycolylneuraminic acid according to the conventional methods which use normal mice such as Balb/c mice as immunized animals. On the other hand, it is known that mice with autoimmune disease produce antibodies against autoantigens such as anti-nuclear antibodies or anti-erythrocyte antibodies (see col., 8, last ¶). The `198 patent teaches that the produced antibodies are very effective for study of cancer's occurrence mechanism diagnosis and treatment (see abstract in particular).

The claimed invention differs from the reference teachings only by the recitation that the mouse is Fas function defects and the antigen is glypican in claim 1 such as glypican-3 in claim 6.

Makino et al teach that comparative studies between male BXSB and MRL/lpr mice at the onset period. Makino et al teach that MRL/lpr mice had much higher level of serum IC than male BXSB mice at 13 weeks as assessed by fluid- and solid-phase C1q-binding assays (see abstract).

Lage et al teach that proteoglycans are glycoproteins containing various sulfated glycosaminoglycan residues, e.g., heparan sulfate. These glycoproteins, designated as heparan sulfate proteoglycans (HSPGs), are widely distributed in many tissues, occurring in various forms of extracellular matrices, at cell surfaces, and in intracellular granules. HS is a regulatory polysaccharide. Glypicans, also called glypican-related integral membrane proteoglycans (GRIPS), are one of two major families of transmembrane HSPGs. The glypicans are anchored to membranes by a glycosyl-phosphatidylinositol (GPI) anchor. This covalently attached GPI residue was the source of the term glypican, which is derived from glycosylphosphatidylinositol-anchored proteoglycan (see page 438, under Introduction). Lage et al teach that despite many

efforts, no mAb specifically recognizing the GPC3 polypeptide could be generated, it appears obvious that GPC3 is only weakly or not immunogenic in mice.

Claim 7 is included because human GPC3 has 94% sequence identity with the mouse.

It would have been obvious to one of ordinary skill in the art at the time the invention was made to make mAb against GPC3 using MRL/lpr mice autoimmune mice. The teachings of Lage et al pertaining to the difficulties in generating mAb specifically recognizing the GP3 polypeptide because GPC3 is only weakly or not immunogenic in mice and the teachings of the `390 publication and `198 patent indicating success in generating specific antibody in the face of having to solve a similar problem would have led one of ordinary skill in the art at the time the invention was made to combine the references to solve a well known problem in the art. The strongest rationale for combining reference is a recognition, expressly or implicitly in the prior art or drawn from a convincing line of reasoning based on established scientific principles or legal precedent that some advantage or expected beneficial result would have been produced by their combination In re Sernaker 17 USPQ 1, 5-6 (Fed. Cir. 1983) see MPEP 2144.

The skilled in the art would be motivated to use MRL/lpr mice because MRL/lpr would produce much higher level of serum IC than male BXSB mice.

From the combined teachings of the references, it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention. Therefore, the invention as a whole was *prima facie* obvious to one of ordinary skill in the art at the time the invention was made, as evidenced by the references, especially in the absence of evidence to the contrary.

Applicant's arguments, filed 6/4/09, have been fully considered, but have not been found convincing.

Applicants submit that the '390 publication and the '198 patent teach away from the instant invention. The '198 patent and the '390 publication disclose that, when N- glycolyneuraminic acid containing glycolipids are used an immunogen, a mouse having an autoimmune disease is preferably used to produce antibodies. However, the '198 patent and the '390 publication further describe that a normal mouse, such as a Balb/c mouse, which has been administered bacterial lipopolysaccharide (LPS), or the like, to enhance the production of autoantibodies, and is in an autoimmune disease state, may also be used as an immunized animal. That is, the ' 198 patent and the '390 publication suggest that either a Balb/c mouse, administered with LPS, or the like, or a mouse having an autoimmune disease, such as an NZB mouse, are equally useful fbr generating antibodies against N-glycolylneuiaminic acid-containing glycolipids, however, none of the cited references teach or suggest that a mouse with Fas function defects, such as an MRL/lpr mouse, is more useful than a Balb/c mouse administered with LPS, or the like. to generate an antibody against a human protein, which has a high percentage of sequence identity with an endogenous protein of an immunized animal. Applicants submit that an ordinary artisan

would have been reluctant to use a mouse having an autoimmune disease to produce the described antibodies if the ordinary artisan was aware at the time of the invention that there is no advantage to using such a mouse when a Balb/c mouse, administered with LPS or the like, is equivalent to a mouse having an autoimmune disease for the generation of antibodies against N-glycolylneuraminic acid-containing glycolipids. Accordingly, the cited references teach away from using mice having an autoimmune disease to generate the described antibodies. Based upon the foregoing, the claims are not obvious in view of the cited references. Accordingly, Applicants respectfully request withdrawal of the rejection.

However, a prior art reference may be considered to teach away when "a person of ordinary skill, upon reading the reference, would be discouraged from following the path set out in the reference, or would be led in a direction divergent from the path that was taken by the applicant." See In re Gurley , 31 USPQ2d 1130, 1131 (Fed. Cir. 1994).

However, neither the '390 publication nor the '198 patent diverges and points in a technical direction away from the present invention. Prior art must be looked at in its entirety. Here in contrast to applicant's assertions of teaching away by the prior art because the preferred embodiments do not constitute a teaching away from a broader disclosure or nonpreferred embodiments. See In re Susi USPQ 423 (CCPA 1971). The instant case, the `198 patent teaches a preparation containing immunogens is used to immunize animals, wherein the animal has autoimmune disease and more preferable a mice with autoimmune disease are the most preferable, wherein the mice with autoimmune disease such as NZB, BXSB and other mice available. The skilled in the art would not have been reluctant to use a mouse having an autoimmune disease to produce the described antibodies with the advantage that the autoimmune disease mouse produce antibodies against autoantigens. With respect to the Balb/c mouse being administered with a polyclonal antibody B cells activator, e.g., LPS, the `198 patent qualifies the use of the PBA induce Balb/c in that if the mice become autoimmune by raising their autoantibody producing ability caused by the injection of LPS or dexstran sulfate (see col., 8, lines 37-53 and claim 12 in particular). The stated advantage of the autoimmune mice is that it produces antibody against less immunogenic glycolipids, which are autoantigens for mice. The `198 patent teaches that it is difficult to obtain monoclonal antibodies specific for glycolipids using conventional methods which use normal mice such as Balc/c mice as immunized animal. On the other hand, it is known that mice with autoimmune disease produce antibodies against autoantigens. Accordingly, there are no teachings away, but rather, the reference provides a clear advantage to use a mice with autoimmune disease to produce antibodies against extremely weak immunogenic determinants such as GPC3.

Regarding the Fas function defects, such as MRL/lpr mouse, Makino et al teach that MRL/lpr mice had much higher level of serum IC than male BXSB mice at 13 weeks as assessed by fluid- and solid-phase C1q-binding assays (see abstract). Accordingly, one skilled in the art at the time the invention was made would find MRL/lpr mouse more useful than BXSB mouse to generate more antibodies in the serum.

11. Claims 1-2, 5-7, 9, 13-14, 16 and 18-21 are rejected under 35 U.S.C. 103(a) as being unpatentable over JP-01047390 (IDS ref. No. BB) OR US. Pat. No. 4,965,198 (IDS AA), each in view of U.S. Pat. No. 5,641,488 and Lage et al (2001, IDS CA).

The teachings of the `390 publication and the `198 patent have been discussed, supra.

The claimed invention differs from the reference teachings only by the recitation that the nonhuman animal that develops the autoimmune disease is Fas function defects in claims 1 and 7, the immunogen is glypican protein in claim 1 or a human native protein which has a sequence identity of 94% or more at the amino acid sequence level to a homolog protein of the mouse to be immunized in claim 7 and the mouse is the MRL/lpr mouse in claim 9.

The `488 patent teaches methods for producing an antibody which specifically binds to a chosen antigen using the so-called autoreactive animals, such as mouse strains NZBXSWR(F1) and MRL lpr/lpr (SLE model) animals may be used. "Autoreactive" animals do not require treatment to undergo B cell hypermutation. Such animals need only be immunized with the immunogen of choice when they are in an autoreactive state. Determination of when the animal is in such a state is easily determined by one skilled in the art (see col. 17, lines 23-30).

Lage et al teach that proteoglycans are glycoproteins containing various sulfated glycosaminoglycan residues, e.g., heparan sulfate. These glycoproteins, designated as heparan sulfate proteoglycans (HSPGs), are widely distributed in many tissues, occurring in various forms of extracellular matrices, at cell surfaces, and in intracellular granules. HS is a regulatory polysaccharide. Glypicans, also called glypican-related integral membrane proteoglycans (GRIPS), are one of two major families of transmembrane HSPGs. The glypicans are anchored to membranes by a glycosyl-phosphatidylinositol (GPI) anchor. This covalently attached GPI residue was the source of the term glypican, which is derived from glycosylphosphatidylinositol-anchored proteoglycan (see page 438, under Introduction). Lage et al teach that despite many efforts, no mAb specifically recognizing the GPC3 polypeptide could be generated, it appears obvious that GPC3 is only weakly or not immunogenic in mice.

Claim 7 is included because human GPC3 has 94% sequence identity with the mouse.

The limitations of claims 13 and 14 are inherent to the MRL/lpr mouse.

It would have been obvious to one of ordinary skill in the art at the time the invention was made to use MRL lpr/lpr taught by the `488 patent in a method for producing an antibody to GPC3. It would have been obvious to one of ordinary skill in the art at the time the invention was made to make mAb against GPC3 using MRL/lpr mice autoimmune mice. The teachings of Lage et al pertaining to the difficulties in generating mAb specifically recognizing the GP3 polypeptide because GPC3 is only weakly or not immunogenic in mice and the teachings of the `390 publication and `198 patent indicating success in generating specific antibody in the face of having to solve a similar problem would have led one of ordinary skill in the art at the time the invention was made to combine the references to solve a well known problem in the art. The

strongest rationale for combining reference is a recognition, expressly or implicitly in the prior art or drawn from a convincing line of reasoning based on established scientific principles or legal precedent that some advantage or expected beneficial result would have been produced by their combination In re Sernaker 17 USPQ 1, 5-6 (Fed. Cir. 1983) see MPEP 2144.

One of ordinary skill in the art at the time the invention was made would have been motivated to do so because such animals need only be immunized with the immunogen of choice when they are in an autoreactive state (i.e., the MRL/lpr mouse need not to be induce with PBA to become autoreactive, spontaneous autoreactive).

From the combined teachings of the references, it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention. Therefore, the invention as a whole was *prima facie* obvious to one of ordinary skill in the art at the time the invention was made, as evidenced by the references, especially in the absence of evidence to the contrary.

Applicant's arguments, filed 6/4/09, have been fully considered, but have not been found convincing.

Applicant continue to argue that the '390 publication and the '198 patent teach away from the instant invention. That is, the cited references do not teach or suggest that a mouse with Fas function defects, such as an MRL/lpr mouse, is more useful than a Balb/c mouse, administered with LPS or the like, for the generation of an antibody against a human protein that has a high percentage of sequence identity with the endogenous protein of an immunized animal. The '488 patent is merely cited for teaching MRI,/lpr animals. Specifically, the '488 patent teaches that "[i]n one embodiment of the invention, so-called autoreactive animals, such as mouse strains NZB×SWR(F1) and MRL Ipr/lpr animals may be used. "Autoreaetive" animals do not require treatment to undergo B cell hyermutation. Such animals need only be immunized with the immunogen of choice when they are in an autoreactive state. Determination of when the animal is in such a state is easily determined by one skilled in the art," see column 17, lines 23-30. Applicants submit that this description does not remedy the deficiencies of the '390 publication, the '198 patent and Lage. Based upon the foregoing, the claims are not obvious over the cited references. Accordingly, Applicants respectfully request withdrawal of the rejection.

However, neither the '390 publication nor the '198 patent diverges and points in a technical direction away from the present invention. Prior art must be looked at in its entirety. Here in contrast to applicant's assertions of teaching away by the prior art because the preferred embodiments do not constitute a teaching away from a broader disclosure or preferred embodiments. See In re Susi USPQ 423 (CCPA 1971). The instant case, the `198 patent teaches a preparation containing immunogens is used to immunize animals, wherein the animal has autoimmune disease and more preferable a mice with autoimmune disease are the most preferable, wherein the mice with autoimmune disease such as NZB, BXSB and other mice available. The skilled in the art would not have been reluctant to use a mouse having an

autoimmune disease to produce the described antibodies with the advantage that the autoimmune disease mice produce antibodies against autoantigens. With respect to the Balb/c mouse being administered with a polyclonal antibody B cells activator, e.g., LPS, the `198 patent qualifies the use of the PBA induce Balb/c in that if the mice become autoimmune by raising their autoantibody producing ability caused by the injection of LPS or dexstran sulfate (see col., 8, lines 37-53 and claim 12 in particular). The stated advantage of the autoimmune mice is that it produces antibody against less immunogenic glycolipids, which are autoantigens for mice. The `198 patent teaches that it is difficult to obtain monoclonal antibodies specific for glycolipids using conventional methods which use normal mice such as Balc/c mice as immunized animal. On the other hand, it is known that mice with autoimmune disease produce antibodies against autoantigens. Accordingly, there are no teachings away, but rather, the reference provides a clear advantage to use a mice with autoimmune disease to produce antibodies against extremely weak immunogenic determinants such as GPC3.

The ` 488 patent provides clear a clear motivation as to why the skilled in the art would use the MRL/lpr mouse because such animals need only be immunized with the immunogen of choice when they are in an autoreactive state (i.e., the MRL/lpr mouse need not to be induce with PBA to become autoreactive, spontaneous autoreactive) compared to the normal mouse Balb/c which require the use of PBA in order to become autoreactive in order to produce autoantibodies.

12. No claim is allowed.

13. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

14. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Maher Haddad whose telephone number is (571) 272-0845. The examiner can normally be reached Monday through Friday from 7:30 am to 4:00 pm. A message may be left on the examiner's voice mail service. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla can be reached on (571) 272-0735. The fax number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

August 12, 2009

/Maher M. Haddad/
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